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COMMUNICATIONS

THE RESPONSE OF THE EYE TO LIGHT IN RELATION TO THE MEASUREMENT OF SUBJECTIVE BRIGHTNESS AND CONTRAST

BY

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Introduction

In recent years it has become increasingly recognised[1] that a satisfactory analysis of certain lighting problems can only be arrived at by evaluating the subjective effects of light. Some method of measuring the magnitude of the visual response is required for various conditions of stimulation and adaptation of the eye. Further, since contrast plays an all-important part in vision, that, too, should be measured, and the factors tending either to increase or decrease it, understood. The object of this paper has therefore been to summarise the information available about those processes which govern the magnitude of the visual response, to indicate how they come into play under normal conditions of seeing, and to put forward suggestions regarding

E.N.T.

suitable methods for measuring subjective brightness and contrast.

For the sake of brevity, at certain points in the paper, data are given in note form.

SECTION I

THE RESPONSE OF THE EYE TO LIGHT

A. Objective Data

RESPONSE IN SINGLE FIBRES OF THE OPTIC NERVE.

During the last few years a technique has been developed by which, when a beam of light stimulates the retina, the impulses from a single element in the retina of certain eyes have been recorded as they travel down the associated nerve fibre in the optic nerve. The methods used vary from one eye to another, but in general an eye is excised from the animal with a portion of the optic nerve attached. The nerve sheath is then removed and the nerve itself frayed out into small bundles. These bundles may then be split into still finer strands if necessary until, as shown by the nerve record, a response from a single fibre has been obtained. The response which is measured is the potential that develops between the cut end of the nerve strand and an uninjured portion of the nerve. Electrical contact is made with suitable electrodes, *e.g.*, silk threads soaked in sea-water, the potential changes are amplified, and the result recorded in an oscillograph. In some cases it is necessary to obtain the impulses from fibres on the anterior surface of the retina before they have reached the optic nerve, in which case the anterior half of the excised eye is removed and the potential between the dissected nerve fibre and the surface of the retina is recorded.

Results.—Typical response obtained from eye of horseshoe crab shown in Fig. 1.

Response consists of series of impulses all of same magnitude. Magnitude varies from fibre to fibre. Short latent period before discharge commences. Initial discharge consists of rapid burst of impulses; frequency falls rapidly at first, approaching steady

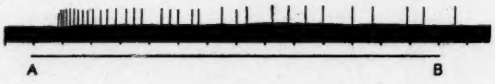

FIG. 1.

Impulses in single nerve fibre from eye of horseshoe crab. (Hartline and Graham[2].)
Line A B shows duration of stimulation Time in fifth of seconds shown below record.

value. With increased intensity of light, latent period shorter, frequency of discharge greater. Range of intensity to which single unit responds may be as great as 1 to 1,000,000.[2]

For short duration of exposure to light stimulus, change in time of exposure is equivalent to change in intensity of stimulus.[3]

Results from cold-blooded vertebrate eyes (*e.g.*, bull-frog) show different types of response: 20 per cent. show response of type in Fig. 1; 50 per cent. show burst of impulses at onset of illumination and another at cessation, but no impulses during steady illumination; 30 per cent. show no impulses at onset or during illumination, but give marked discharge when light cut off.[4]

With the scallop, two kinds of sense cell observed; one responds at the onset and during illumination, the other to cessation of illumination.[5]

RETINAL ACTION POTENTIAL.

Instead of obtaining records from individual fibres, an alternative technique has been developed from which a composite curve can be obtained showing the potential that is developed over a more extended area of the retina. Again various methods are available

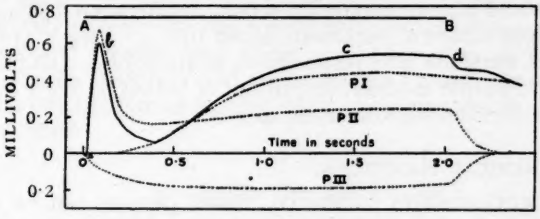

FIG. 2.

Analysis of retinal action potential of dark-adapted cat's eye. (Granit[6].)
Line A B shows duration of stimulation of intensity 14 ml. Resultant response shown by continuous line. Individual components of response, P I, P II, and P III shown by dotted curves.

of deriving this so-called retinal action potential, but a typical way is to place an excised eye in a holder in which contacts can be made to the cornea and the back of the eye by means of suitable electrodes. The potentials which develop when light enters the eye are amplified as before and recorded in an oscillograph. An alternative process is to use a decerebrate animal, in which case contact is made with the cornea and the decerebration wound.

Results.—Typical record is shown in Fig. 2.

Electroretinogram analysed by Granit[6] into three components, PI, PII, PIII. PIII is negative; on stimulation, commences

before positive PII to produce negative notch a. Potential rises rapidly to maximum, b, and then falls. Steady positive rise c, follows. At cessation of stimulation, off-effect, d, occurs, explained as interference construction between positive and negative components. Components PI, PII, PIII can be isolated by suitable treatment, *e.g.*, PII removed from retinogram of cat by asphyxia.

PI not known to be connected with discharge in optic nerve. PII corresponds to excitation, PIII to inhibition.

Light adaptation increases inhibitory component PIII[7]. Light adapted eye shows brisk response, dark adapted eye is sluggish.

Interaction between separated areas of retina demonstrated by effect on latent period of stimulating retina in separated areas.[6, 8]

Stimulation of retina by bright light can amplify difference between responses of two test lights viewed after primary stimulation.[9]

Prolonged light adaptation reduces size of b-wave obtained from test light. During subsequent dark adaptation, size of b-wave returns gradually to normal.[10]

Rod retina (*e.g.*, albino rat) has maximum of sensitivity for equal energy spectrum between 0·50 and 0·51 μ. When eye exposed to bright sunshine, no response at all. With dark-adapted frog's eye (rods and cones), maximum sensitivity between 0·50 and 0·51 μ. When light adapted, maximum about 0·56 μ. Frog's eye exposed to bright sunshine still responds to stimulation. Conclusion that rod-visual purple mechanism put out of action by high intensities, but cone mechanism continues to react.[11]

PHOTOCHEMICAL REACTIONS.

The light sensitive substance, visual purple, can be extracted from various retinae by the appropriate chemical treatment (see, *e.g.*, Lythgoe[12]). Measurement of the spectral absorption curve of bleached and unbleached visual purple can be made, the bleaching process can be followed, and although no appreciable regeneration of the substance after bleaching can be observed once it has been extracted from the eye, the regeneration has been determined by measuring the concentration of visual purple extracted from the eyes of rats at intervals after complete bleaching.

Results.—After allowance for impurities, maximum absorption of unbleached visual purple at about 0·50 μ. Intermediate product is formed in course of bleaching; orange colour of this product depends on temperature and pH of solution. Not certain whether this substance sensitive to light.[12]

Regeneration of visual purple in rat eye (rods) slow process continuing for several hours.[13]

Amount of bleaching governed by intensity of incident light.

No certain information available of photochemical substances in cone retina.

IRIS.

As is well known, the rapid enlargement and contraction of the iris serves to compensate for sudden changes in the illumination level. A given diameter of iris is not uniquely related to one level of illumination. An illumination that appears bright to a dark-adapted eye, and therefore, produces a contraction of the iris, may appear dark to a light-adapted eye, thus causing the iris to expand. The average range in the diameter of the iris is from 2 mm. to 8 mm. The speed of reaction of the iris, a completely reflex action, varies from one person to another, but the adjustment is usually complete in $\frac{1}{2}$ sec. or thereabouts.

ANATOMY OF THE EYE.

Since the response from rods and cones is very different, their distribution in the retina is a relevant factor in an analysis of the subjective effects of light. Histological examination of the retina reveals two types of neurone, the rod and cone. The anatomical distinction is frequently not very marked, but the rods, which are held responsible for vision at low illumination (scotopic mechanism), can be defined as the neurones secreting visual purple.[11] In the human retina, some 120,000,000 rods are found and 6,500,000 cones. In the fovea, the seat of distinct vision, only cones are present, and these are believed to show a 1:1 relation between receptors and nerve fibres. In outer parts of the retina the rods predominate, and many rods may converge on to one nerve fibre.

B. Subjective Data

THRESHOLDS.

Subjective measurements of the visual response are difficult. The inherent obstacle is the impossibility of measuring sensations, and the method most commonly employed to avoid this difficulty has been to measure the intensity of a patch of light when it just becomes visible. On the assumption that the threshold sensation always requires the same visual response to elicit it, measurement of the threshold intensity does provide a means of recording the state of sensitivity of the eye under various conditions of adaptation, etc. This assumption is reasonable, at least for restricted conditions of observation, *e.g.*, given angular size of threshold patch. On the other hand, to derive anything about the magnitude of response that would result from a stimulus well above the threshold intensity necessitates assumptions concerning the retinal processes that are difficult to justify. For this reason extensive deductions from threshold measurements for conditions above the threshold must be accepted with reserve.

Results.—Dark adaptation: process observed by measuring threshold intensity at various times after observer placed in dark following pre-stimulation to definite intensity. Curve obtained depends on intensity of initial stimulus, area of retina used to view threshold patch. Typical result, Fig. 3.[14] Where curve has cusp, initial part presumably represents regeneration of cone photochemical substance, later part regeneration of visual purple. Where no cusp, only visual purple involved.

Retinal variation of sensitivity: for light-adapted eye, maximum sensitivity at fovea and rapid falling off in periphery;[15] for dark adapted eye, minimum sensitivity at fovea, maximum 10° or 15° from fovea.

Glare: measurements of threshold intensity viewed simultaneously with glare source provide record of loss of sensitivity due to glare. Sensitivity, β, shown experimentally[17, 18] to obey law of type:—

$$\beta = \kappa \frac{E}{\theta^2}$$

where κ is a constant, E illumination produced by glare source in plane of observer's eye, and κ is angle between test patch and glare source. Formula as given applies when test patch viewed against black background.

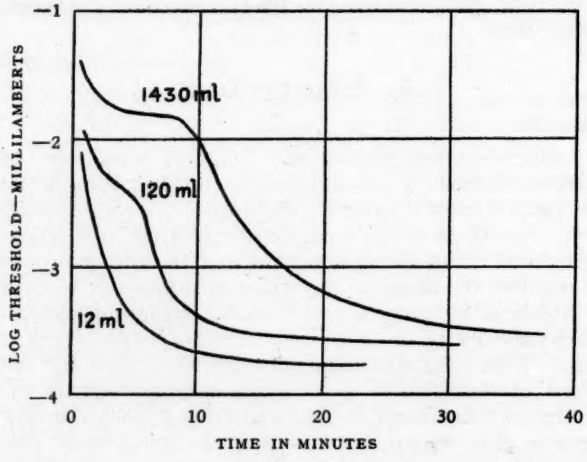

FIG. 3.

Dark adaptation process for various intensities of previous stimulation. (Winsor and Clark[14].)
Initial intensity of light adaptation shown in diagram.

Light adaptation: threshold measurements for various intensities of light adaptation show lowering of sensitivity as intensity increased, Fig. 4.[19]

Brightness discrimination: sensitivity to brightness differences recorded by measurement of just noticeable brightness difference, $\triangle$ B, at various intensities, B.

Relation between B and $\triangle$ B shown in Fig. 5.[20]

Result affected if eye maintained at given adaptation level instead of adapting to test field. Relation between B and $\triangle$B for constant adaptation shown in Fig. 6.[20]

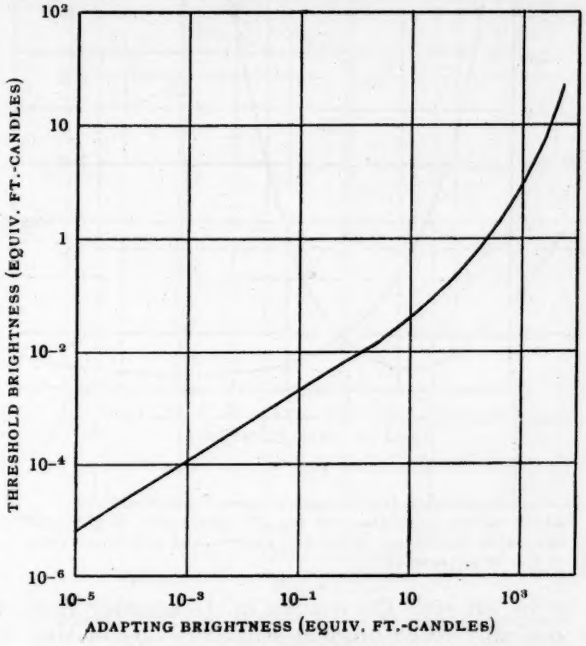

FIG. 4.

Threshold brightness for various degrees of light adaptation. (Nutting[19].)

DEVELOPMENT OF RESPONSE.

The visual response takes a finite, but very short, time to develop, and having reached a maximum value, the response diminishes. Broca and Sulzer[21] and others have measured the apparent brightness of a flashing light for various short exposures by comparison with a fixed light. The development of the response is shown in Fig. 7, as obtained recently by Schouten,[22] by the method of binocular matching.

LIGHT ADAPTATION.

Apart from measurements of threshold intensities at various adaptation levels, little has been recorded of the light adaptation process except for the results obtained recently by the binocular matching method.[22, 23, 24] In this method the right eye views a test patch and the left eye a comparison patch, and the two may be equated both for colour and intensity. The right eye is then subjected to various degrees of light adaptation, and the changes induced are measured by comparison and equation with the patch

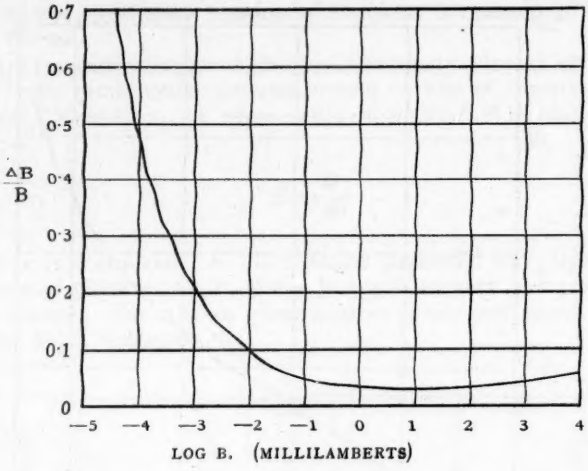

FIG. 5.

Brightness discrimination curve. (Abribat[20].)
ΔB/B shown in relation to log B, where ΔB is the just noticeable brightness difference observed at different values of the brightness B.

viewed in the left eye. On removal of the adapting field, the eye returns gradually to its original sensitivity; by making a rapid series of matches, the course of this recovery can be derived. The author's results have been confined to the fovea and presumably record the photochemical reaction that takes place there; Schouten has included the effect of extra-foveal adaptation, and has also investigated a more rapid action of the adapting light.

Results.—Within limits, depression of sensitivity approximately proportional to adaptation intensity. For adaptation of 6,000 photons. mean result for 85 observers gave reduction of sensitivity by factor 12·5. (A photon is the unit of retinal illumination. An illumination of one photon is obtained when a surface having a brightness of 1 cdl. per sq. metre is viewed through a pupil

area of 1 sq. mm.) Direct linear recovery for some observers after white adaptation, delayed recovery for others, Fig. 8.[25] Recovery effectively complete after two or three minutes. Irregular recovery curves obtained after very high intensity adaptations.[23] Variety of effects obtained after coloured adaptations, which cannot conveniently be summarised here.[23, 24] Depression of sensitivity for

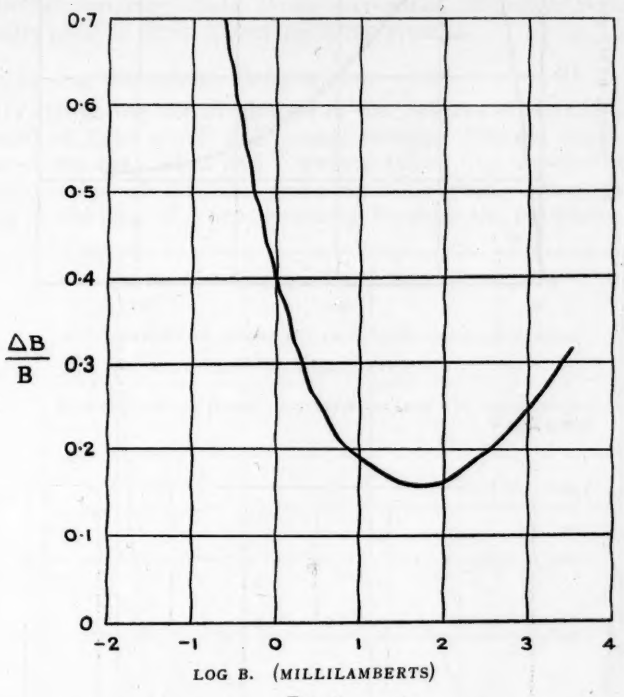

FIG. 6.

Brightness discrimination curve for eye maintained at constant state of adaptation to field of brightness 3·8 cdls. per sq. ft. (Abribat[20].)

given adaptation increases with time of exposure until approximately steady state reached after about one minute, Fig. 9.

When sensitivity tested after very short exposure to adapting light[22] some depression of sensitivity found already, which increases with longer (but still very short) exposures until steady state reached, Fig. 10. Prolonged exposure caused the further depression already illustrated in Fig. 9. Rapid adaptation called α-adaptation, slower process β-adaptation (Schouten). Exposure to glare source (viewed 6° from fovea) giving illumination at

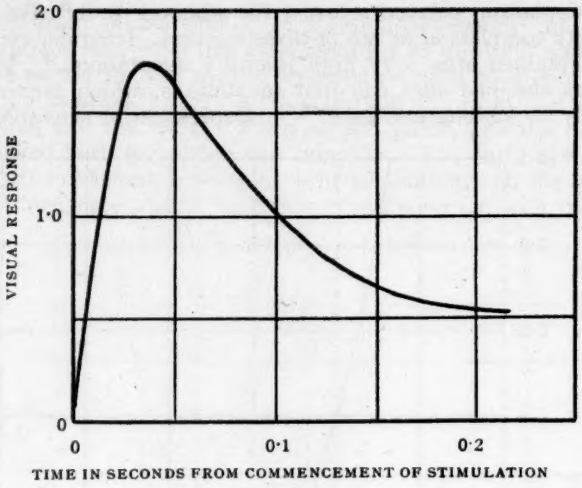

FIG. 7

Development of visual response immediately eye is stimulated. (Schouten[22].)

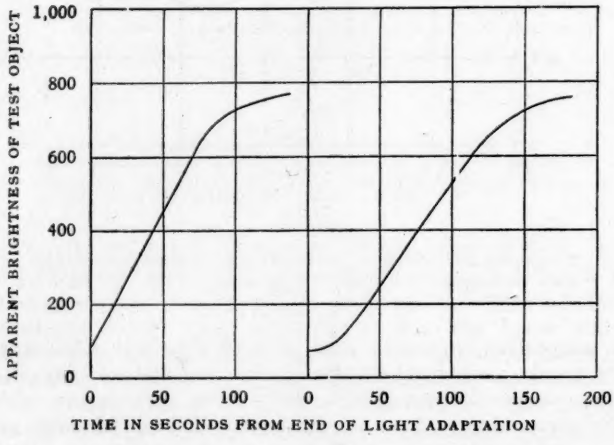

FIG. 8.

Recovery of sensitivity of fovea after adaptation to intensity of 6,000 photons. Two types of recovery curve. (Wright[25].)

observer's eye of 17 ft. c. lowered sensitivity of retina at fovea to one-eighth initial value. Increase of adaptation intensity causes increased *a*-effect. *a*-adaptation affects adjoining parts of retina in addition to area actually stimulated.[22] β-adaptation local adaptation confined to illuminated portion of retina.[24] Time of recovery from *a*-adaptation uncertain. If electrical effect as believed, recovery likely to be very rapid, although Schouten's results tend to show slower recovery process.

STILES AND CRAWFORD EFFECT.

By comparing the brightness of two patches formed by narrow pencils of light which had passed through different parts of the lens of the eye, Stiles and Crawford found that a pencil passing centrally through the lens gave a markedly greater visual response than in the case of a pencil passing through the periphery of the

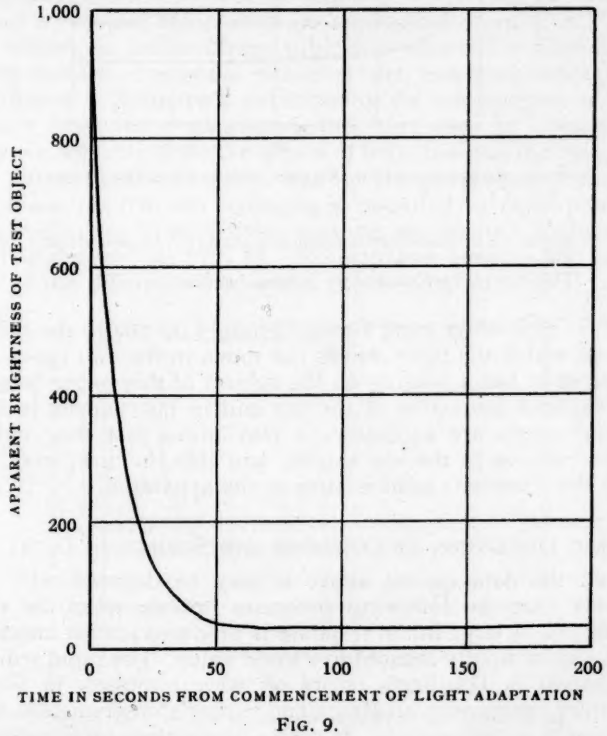

FIG. 9.

Reduction of sensitivity of fovea during process of light adaptation. (Wright[23].)
This records photo-chemical or β-adaptation process.

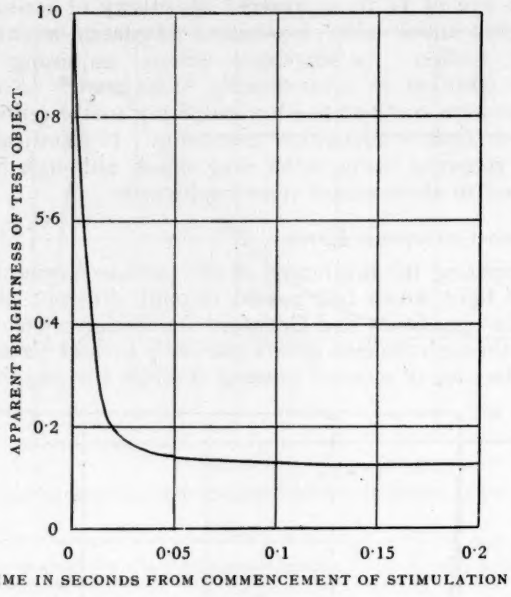

FIG. 10.

Reduction of sensitivity of fovea immediately eye is stimulated. (Schouten[22].)
This records electrical or *a*— adaptation process.

lens.[26, 27] This effect must almost certainly be due to the different angle at which the light strikes the retina in the two cases. The phenomenon has a bearing on the subject of this paper inasmuch as it modifies the action of the iris and in instruments in which artificial pupils are necessary, it may mean that they must be centred relative to the eye pupils, and this, in turn, may mean fixing the observer's head relative to the apparatus.

GENERAL DISCUSSION OF OBJECTIVE AND SUBJECTIVE DATA.

From the data quoted above it may be deduced with some certainty that the following processes operate when the eye is stimulated. A large initial response is produced almost instantaneously, but is rapidly reduced to a lower value. The rapid reduction is apparent in Hartline's record of nerve impulses, in Granit's inhibitory component, in Broca and Sulzer's observations, and in Schouten's *a*—adaptation. It seems likely also, as suggested by Schouten, that this rapid reaction is identical with the effect of simultaneous contrast by which, as is well known, a surround

field or a glare source may produce a profound effect on the apparent brightness and colour of a test field. The diameter of the iris will also rapidly change when the stimulus is introduced. On continued exposure to the stimulus, a photo-chemical decomposition occurs and continues until a balance between decomposition and regeneration is reached within a minute or so. If the intensity is high, the visual purple system in the rods will be put out of action, but cone vision will continue to operate, although at a low sensitivity level. These facts follow from the subjective adaptation phenomena, Granit's measurements on frog's eyes, and experiments with visual purple. Removal of the stimulus causes a partial rapid recovery of sensitivity due to the removal of the inhibitory component or *a*—adaptation, and also sets in motion a more gradual recovery due to photochemical regeneration taking place. In the cones this regeneration is more or less complete in two or three minutes, in the rods at least half an hour is required before recovery approaches completion. These facts follow, as before, from subjective adaptation phenomena, from retinal action potential measurements, and from visual purple experiments. Schouten's experiments do not suggest a rapid recovery from the *a*-adaptation, but it is easy to demonstrate that some recovery from the effects of simultaneous contrast takes place almost instantaneously, and for the purpose of this paper we will assume that the *a*-process is essentially a rapid process.

These changes in sensitivity may be represented qualitatively in a diagram such as Fig. 11. No emphasis need be laid on the shape of the different components of the diagram, but it can be

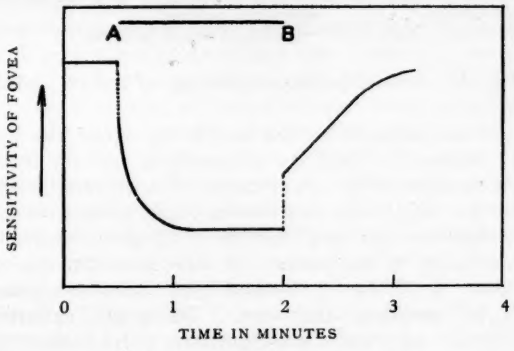

FIG. 11.

Diagram to illustrate change of sensitivity of fovea during and after stimulation.
Line A B shows duration of stimulation.
Dotted part of curve represents rapid, electrical process.
Continuous part of curve represents photo-chemical process.

regarded as giving a general indication of what is taking place. The visual response, which is assumed to be proportional to the sensitivity, will follow a curve similar to that of Fig. 11.

Consider next what happens when one patch of light is viewed at the fovea and a second patch is thrown on a neighbouring part of the retina. The second patch will tend to inhibit or suppress the response from the first patch; the sensitivity curve will then

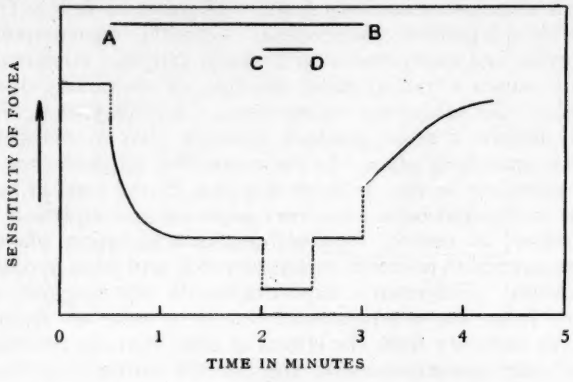

FIG. 12.

Diagram to illustrate change of sensitivity of fovea during and after stimulation, including effect of glare source imaged on neighbouring part of retina.
Line A B shows duration of stimulation at fovea.
Line C D shows duration of stimulation from glare source.
Dotted part . . . . . electrical process.
Continuous part ——— photo-chemical process.

be as in Fig. 12, for the particular timing of the two patches given in the diagram.

Finally, what happens to the sensitivity when the eye views a scene, as it normally does, by scanning it rapidly from point to point, each element of the scene being of a different intensity from its neighbour? Since the eye moves rapidly over the scene there will be no time for any local variations of photo-chemical concentration to develop in the retina; in others words, the state of the β-adaptation will be governed by the integrated value of the light entering the eye. To some extent this will also be true of the α-adaptation; for the contrasting action between one light and another is greater the nearer they are to one another, and to that extent the α-process is a local process. At the same time it is clear, especially from Schouten's work, that the reaction taking place on one retinal area may spread over a considerable fraction of the retina. It is therefore

desirable to separate the *a*—adaptation into two parts, one a local and the other a general effect. This division is not essential and the boundary between the two effects cannot be clearly demarcated. For our purpose we may regard the general *a*—effect as governed by an integrated value of the light from the scene and assume that it will have a roughly constant value for any given scene, while the local effect will vary rapidly as the eye darts about over the scene.

Summarising, we can say that the sensitivity of any point of the retina is governed by

(1) the β-adaptation, which will be a function of the integrated light from the scene;

(2) the general component of the *a*-adaptation, which will also be a function of the integrated light from the scene;

(3) the local component of the *a*-adaptation, which will be a function of the illumination at, and surrounding, the point of the retina being considered.

The visual response will then be derived as the product of the sensitivity as defined above and the stimulus acting at the point of the retina concerned.

SECTION II

THE MEASUREMENT OF SUBJECTIVE BRIGHTNESS

Introduction

True measurement of subjective brightness implies the measurement of sensations. This is an impossible process for reasons which the author has discussed elsewhere,[28] and into which it is not proposed to enter here. Yet it is obviously possible to compare the apparent brightness of an object seen under one set of adaptation conditions with another object seen under a different adaptation level; for instance, an approximate comparison or match might be made by memory, or by the method of binocular matching to be described. This comparison does not really measure subjective brightness, but it does make it possible to erect a reference scale of brightnesses. For many purposes this may be all that is required; thus, if we had a standard level of adaptation, we might be able to say that an object in some scene had the same apparent brightness as intensity Y viewed at the standard level. The value Y would then define the brightness of the object, and from previous investigations we might know that such a value was too low for the task in hand. Further, the measurement of brightness is an intermediate stage in the measurement of contrast, since from the reference scale of brightnesses at the standard level, a scale of contrasts can be derived (see Section III); hence a knowledge of the " standard brightness "

of two objects will lead to a determination of the contrast between them.

It is with the measurement of subjective brightness in this sense that this section deals. The problem can be divided into two fairly well-defined parts: (1) To devise a means of viewing various intensities at a standard level of adaptation; and (2) to develop a method of comparing the standard brightness with the unknown brightness that we wish to measure. Superficially, the former appears an easy problem to solve, but this is not altogether true.

Maintenance of a Standard State of Adaptation

The most obvious method would be to adapt the eye to a standard illumination for a definite time, then remove the adapting field rapidly and substitute whatever matching intensity was required. This intensity is then, it seems, being viewed at a standard level

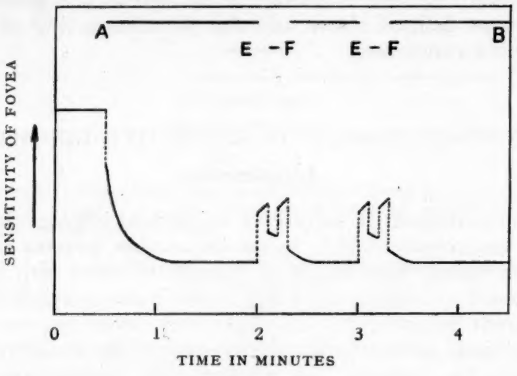

FIG. 13.

Sensitivity changes of fovea when viewing test patch after adaptation to standard intensity.
Line A B shows duration of exposure to standard adaptation.
Line E F shows duration of exposure of test patch.
Dotted part . . . . . electrical process.
Continuous part ——— photo-chemical process.

of adaptation. The error in this assumption can be shown by reference to Fig. 13. Just before the adapting light is removed the sensitivity reaches a standard level, but as soon as it is removed the a-component recovers. On introducing the matching intensity, a new a-component will immediately be introduced, and, if viewed for any time, a new β-process would also come into operation. The instant at which the match is made will

determine the actual sensitivity level of the eye, but it will be seen that, to some extent at least, this level will be governed by the value of the matching intensity required. When measuring a high value of subjective brightness, a high matching intensity will be needed, producing a greater adapting action. Hence strictly it is impossible to view a range of matching intensities all at one standard adaptation level.

Fortunately, although it is a point that requires thorough examination, it is more of academic than practical importance. What is required is a standard technique for reproducing any apparent brightness rather than a standardised state of adaptation. This can be achieved by standardising the following quantities:

(1) The intensity of the preliminary adaptation.
(2) The size of field of this adaptation.
(3) The duration of the adaptation.
(4) The period to elapse between the end of the adaptation and the comparison between the matching intensity and the unknown intensity.
(5) The size of field of the matching intensity.

Method of Comparing Apparent Brightnesses

Assuming that there is available a method of producing any apparent brightness under the appropriate specified conditions, what method is to be used to equate that brightness to the one that has to be measured? Two possible methods seem to be open. The first is that of a memory match in which the test object is viewed first and then matched as nearly as can be remembered against the standard field viewed subsequently; but it will immediately be obvious that any measurement of this sort must be of a low order of accuracy. Even so, it would be a mistake to despise the value of a memory match, and it should undoubtedly be given a thorough trial.

The second method is that of binocular matching in which one eye views the road surface while the other views a comparison patch under standard conditions. A direct match between the two would then be made. This method has been employed successfully during the past few years[22, 23, 24] in the investigation of visual processes, and the author is satisfied as to the validity of the principles involved. These have been discussed in detail in another paper,[25] and space need not be taken here in repeating that discussion. It is easy, however, to visualise a number of fresh difficulties that would follow any attempt to transform the apparatus and method from a laboratory process into an engineering device suitable for use in the field.

One obstacle to measuring subjective brightness that applies to whatever method is contemplated is peculiarly subtle. In Section I

the factors that govern the magnitude of the visual response have been discussed, and reference was made to the fact that, in looking at any scene, the photo-chemical adaptation level will be governed by the integrated light from the scene. But this does not apply when the eye, instead of scanning rapidly across the scene, gazes steadily at a given area for any length of time. Under these conditions local adaptation levels develop on the retina, and in consequence the sensitivity, and hence the visual response, is modified.

From this it follows that any attempt to measure the subjective brightness of part of a scene will be successful provided it is glanced at only intermittently. If the observer looks continuously at what he is trying to measure, his measurement will be erroneous. For faint objects, the error is not likely to be great provided it is not viewed steadily for too long. For bright objects, the error may be considerable.

SECTION III

THE SPECIFICATION OF CONTRAST

Introduction

Although the measurement of sensations is impossible, it is possible to erect a scale of sensation intervals or contrast steps; thus, if two intensities A and B are selected, a third intermediate intensity, C, can be found such that the contrast between A and C is equal to that between C and B. Extending the process over the whole gamut of intensities, it is at least theoretically possible to divide the complete sensation interval from subjective black to blinding white into a large number of equal contrast steps. Supposing, then, that it was required to find the contrast between an object in a road and the background against which it is viewed, it would be necessary to determine the number of contrast steps covered by the interval between the brightnesses of the object and its background. This section, therefore, deals with the method to be followed to derive the contrast scale and the means to adopt to equate an unknown contrast to the appropriate interval on that scale.

The Contrast Scale

From what has already been said earlier, it is obvious that the specification of two intensities A and B is not sufficient to define the subjective contrast between them. This will vary according to the state of adaptation of the eye, and a standard scale must therefore be erected under properly defined conditions of viewing. The obvious choice of conditions would be the same as those

adopted for measuring subjective brightness, and for those conditions, as wide a range of test intensities as possible should be divided into equal contrast steps.

This is not likely to be easy, but it should be possible. Difficulties similar to those referred to in connection with subjective brightness would be encountered, and no doubt others would also arise.

The only data available, as far as the author is aware, which aim at deriving a contrast scale under properly controlled adaptation conditions, are those of Abribat.[20] The one objection to Abribat's work is that the scale is derived from observations of just noticeable differences, a process that may not be identical with the derivation of equal steps of contrast. Space will not

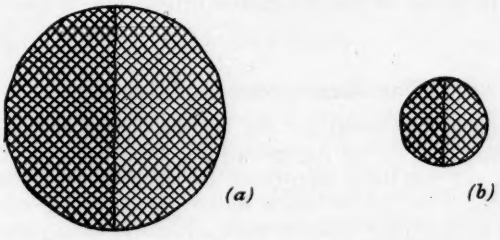

FIG. 14.

Effect of variation of field size on contrast.

permit any detailed discussion of this question (see[28] for a further analysis), but one illustration may serve to indicate the dilemma that might arise from the use of threshold steps.

Suppose the field of view is circular and is divided into two equal parts as in Fig. 14 (*a*), one half being appreciably darker than the other. An observer is conscious of a certain amount of contrast between the two halves. Now suppose the field is reduced in size, Fig. 14 (*b*), without any change in intensity of the patches; the contrast between them will remain practically the same, at least until the field is made very small. This is precisely as expected if contrast were to be evaluated from values of the subjective brightness of the two fields, since these will be practically unaffected by the reduction in size of field. But if the contrast had been calculated from the number of just noticeable steps, the value would have been greater with the large field than with the small field, since the eye is more sensitive to small differences the larger the field of view. How is this discrepancy to be resolved? Is it right to assume that the contrast between the two parts of the field when set for a just noticeable difference is smaller for

the large field than for the small field? Or is the summation of small differences to give a measure of a finite degree of contrast not a legitimate process? In either case the validity of a contrast scale based on just noticeable differences is very uncertain. It must, on the other hand, be admitted that, inversely, the scale derived from finite steps is equally uncertain when applied to very small intensity differences.

A similar discrepancy would probably arise if the time of viewing were reduced to a very short interval. However, as a problem in illuminating engineering, the measurement of contrast is most frequently related to the evaluation of visibility; the specification of contrast would form one element in the specification of visibility, while area and time of exposure could be regarded as separate elements. Regarded in this way, contrast could then be safely measured in terms of the subjective brightness of the contrasting areas.

The Measurement of Contrast

If the view expressed in the last sentence is accepted, the measurement of contrast reduces to the measurement of subjective brightness on the lines suggested in Section II. The measurements would have to be made under the working conditions in which the contrasting objects were normally seen, and recorded, presumably by the binocular method, in terms of the brightness of test patches viewed under standard conditions of adaptation. Interpolation on the standard contrast scale would then give the required value of the contrast.

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THE MECHANISM OF DARK ADAPTATION

A Critical Resumé

BY

R. J. LYTHGOE

IT is a familiar experience that the eyes take some minutes or even hours to accustom themselves to a very low illumination after having been exposed to a high one. Few realise that the increase in sensitivity may be 10,000 fold. If, for example, an illumination of 0·1 metre candle (m.c.) is just visible immediately after switching off a very bright light, an hour or so later in the dark the necessary illumination may have fallen to 0·00001 m.c. Not all scientists working on this and related subjects realize that the process continues for at least an hour, and, according to Achmatov (1927) for at least 24 hours. To measure the least visible illumination of a test surface at various times after going into the dark seems a simple problem, but there are many complicating factors (Phillips, 1939) which have served as traps for the unwary. In this paper, however, we shall not criticize the technique of measurements, but test the adequacy of the physiological theories used to explain the results. The subject now concerns not only a few specialists but also workers from other fields, since it has become generally realized that deficiency of vitamin A may produce faulty dark adaptation.

In times of peace town dwellers never need to use their eyes at the low illuminations encountered in the country at night. Owing to the "black-outs" and the increase in night operations in the services it will not be possible to delay for very much longer a serious study of the problems.

Dual mechanism of dark adaptation.—The curve in Fig. 1 shows the change in visual threshold with time in the dark measured from the time when the observer had been plunged into darkness. He had previously looked at a brightly illuminated

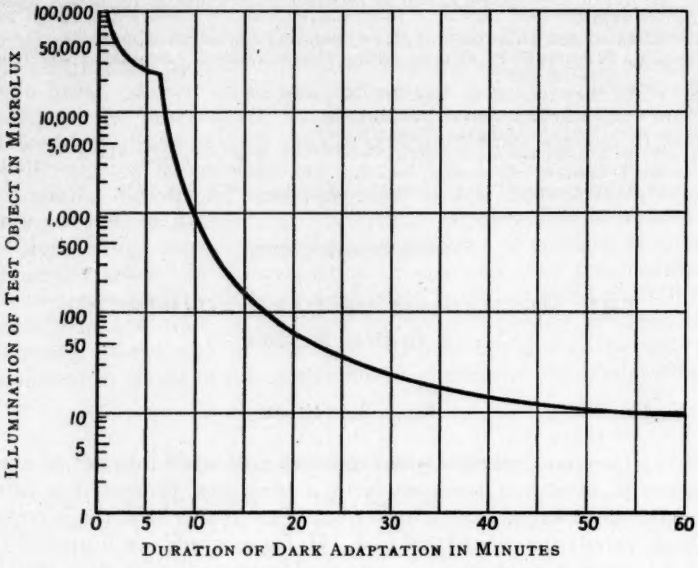

FIG. 1.

Horizontally: Duration of dark adaptation in minutes. Vertically: The smallest visible illumination in microlux (1,000,000 microlux= 1 m.c.). (After Kohlrausch, *Handb. norm. Path. Physiol.*, 1931; see Wright and Granit, *Brit. Jl. Ophthal.*, Monograph Supplement IX, 1938.)

screen for some minutes. It will be seen that the smallest detectable illumination became progressively less during the 60 min. of the test. With a higher initial light adaptation and with greater speed in taking the first reading, a greater increase in sensitivity could have been obtained. The important point to notice is that the curve is divided into two parts which form a kink at their intersection. The dual nature of the curve suggests that two mechanisms are involved and that they adapt at different rates. (Kohlrausch, 1922, Hecht, 1929.) The generally accepted explanation is that the first part of the curve, lasting 6-11 min., reveals the adaptation of the photopic mechanism of vision, which is mediated by the retinal cones. The second part of the curve reveals the adaptation of the scotopic mechanism, which is mediated by the retinal rods and their visual purple. The scotopic mechanism, although capable of a very great increase in sensitivity, seems to exhibit an initial delay in adaptation. As a result, only test-patch illuminations which are sufficiently high to stimulate photopic vision can be seen during the early stage of

adaptation. Photopic vision is completely adapted in about 6 min. (Hecht, 1921), and a little later, scotopic vision becomes sufficiently sensitive to appreciate illuminations which are now too low to stimulate photopic vision. This view is based on the alteration in the relative prominence of one or other part of the curve when the experiment is conducted with light of different wave-lengths, and with observation by different parts of the retina (Kohlrausch, 1922, Hecht, 1937).

Our belief in a dual mechanism of vision is also based on experiments in which some visual function, such as visual acuity, is measured over a wide range of illuminations. As the illumination is raised from a low value, visual acuity improves at one rate up to a certain illumination. Up to this point scotopic vision is being stimulated. Beyond it visual acuity improves at another rate corresponding to the activity of the photopic mechanism (König, 1897, Shlaer, 1937).

Duplicity Theory.—This theory states that photopic and scotopic vision are mediated by the cones and the rods respectively. It is based on a large amount of circumstantial evidence. As an example we may mention the similarity between the phenomena of scotopic vision and the properties of visual purple. This substance, which is found only in the rods, can be brought into solution, and its properties can be accurately investigated. It is found, for instance, that wave-lengths around 502 mμ appear the brightest in scotopic vision, that is, when the spectrum looked at is of low intensity. This wave-length is also that at which visual purple absorbs most of the incident light (König, 1894, Dartnall and Goodeve, 1937, Lythgoe, 1937), and it is also the wave-length which is most capable of bleaching visual purple (Trendelenburg, 1904, Schneider, Goodeve and Lythgoe, 1939). When the spectrum is brighter, maximum luminosity is at about 555 mμ, but we cannot explain photopic vision, which is now being stimulated, in terms of the properties of any known substance in the retina.

There are very few visual judgments which show marked changes both during adaptation to darkness and also as a result of increasing the illumination. The perception of flicker is an exception, since it is peculiarly sensitive to changes in both these conditions. Above about 0·1 m.c. the critical frequency of flicker varies approximately as the logarithm of the illumination (Porter, 1902, etc.). Suppose we have a small test-patch in which darkness and an illumination of 1·48 m.c. alternate for equal intervals of time. If we increase the rate of alternation, all sensation of flicker will vanish at 19·2 flashes per sec. If now we use ten times the brightness, 30·1 (19·2 + 10·9) flashes a second will be necessary to abolish flicker, whilst if we again increase it ten fold, 41·0 (30·1

+ 10·9) flashes will be necessary. This applies only to illuminations above 0·1 – 0·2 m.c. Below this value scotopic vision is in action and a quite different relation holds (Porter, 1902, Lythgoe and Tansley, 1929*a*, Hecht and Verrijp, 1933). We may notice in passing that there is no scotopic component when the relation is investigated by red light longer than a certain wave-length. This observation is in conformity with the fact that this wave-length is not absorbed by, nor does it bleach visual purple. The distinction between scotopic and photopic vision is most marked with a blue of short wave-length.

The behaviour of the critical frequency of flicker during dark adaptation is best investigated by measuring the fusion frequency whilst the illumination of the flash is kept constant. This method gives us a fresh means of studying dark adaptation. Normally we measure the least perceptible brightness at different times, first the photopic and then the scotopic mechanism being stimulated. In the experiments with flicker, however, the brightness can be maintained at such a level as to stimulate the photopic mechanism or only the scotopic mechanism throughout dark adaptation. In a typical experiment the observer would sit inside a whitened cube with brightly lighted walls. Through a small hole (1°) in the cube he would see the flashing test-patch and he could then increase the rate of alternation of light and dark until the test-patch ceased to flicker. The light in the cube would then be extinguished, and the observer would repeat the setting at regular intervals, during which period he would see the small illuminated test-patch in dark surroundings. For illuminations which are too low to stimulate any but the scotopic mechanism, the fusion frequency rises during dark adaptation (Fig. 2A). For illuminations high enough to stimulate the photopic mechanism, the fusion frequency actually falls during dark adaptation, showing that the eye is becoming progressively less sensitive to flicker. Here for the first time we meet a visual judgment which is performed less well by the dark-adapted eye. For intermediate illuminations of the test-patch there is a fall followed by a rise in the critical frequency (Lythgoe and Tansley, 1929*a*), revealing the dual nature of dark adaptation. These experiments show that dark adaptation is not a process manifested only by a progressive lowering of the light threshold, but that throughout its course there is some fundamental change which causes the perceptions of both scotopic and photopic vision to be modified.

For the sake of completeness we may mention here that there is an area in the centre of the retina subtending about 3° where there are no rods and where the phenomena of scotopic vision are absent. Peripheral to this area the density of the cone population

falls off, but that of the rods increases up to a ring of retina which subtends an angle of about 20° to the fixation point, whence it falls off slowly. As is to be expected, the photopic parts of our curves become progressively less evident, and, in general, the contributions of the scotopic portion increases with the increase in rods (Lythgoe and Tansley, 1929*b*, p. 87, l. 22, Hecht, 1937, p. 262, l. 1).

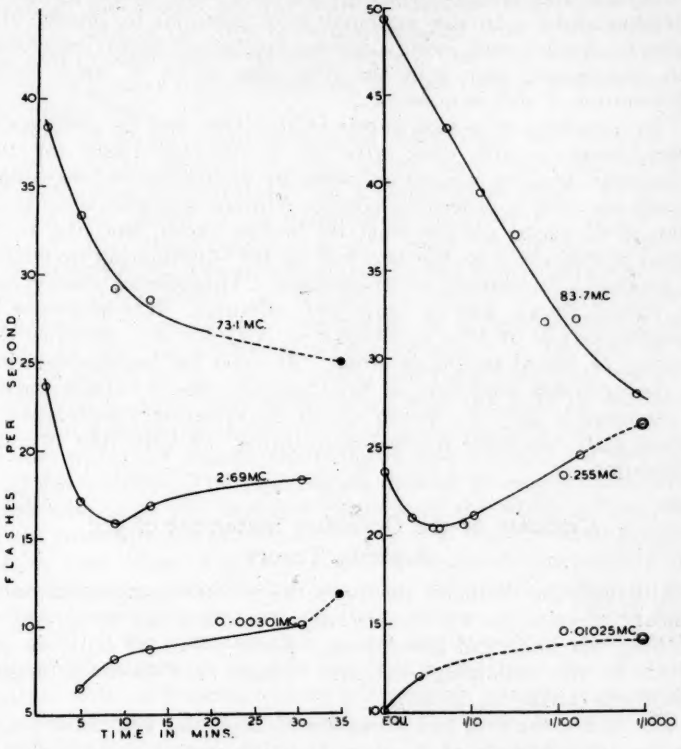

FIG. 2A. FIG. 2B.

To show the similarity between the changes in the critical frequency of flicker (1) during dark adaptation (figure to left) and (2) as a result of changing the brightness of the field surrounding the test-patch (figure to the right). For any one curve the illumination of the test-patch was unaltered (see values set against each curve). All observations were made with 10° of eccentric vision. In the figure to the right the values equ. 1/10, 1/100, etc., refer to conditions where the surrounding field of vision was equally bright, one-tenth as bright and one-hundredth as bright as the test-patch, respectively. ● After 60 mins. in the dark. ◎ Surrounding field of vision completely dark.

The rôle of visual purple in dark adaptation.—Any animal which has rods will be found to have visual purple in its retina (in the rods) if the animal has been kept in the dark for an hour or so before the retina is removed. On bringing the retina to the light the visual purple will be bleached, and instead of being a bluish-red, it will become orange, yellow or almost colourless according to the conditions of the experiment. If the bleached retina of a frog is again put into the dark some visual purple is "regenerated." In the mammal it is essential to replace the retina in contact with living pigment epithelium before regeneration will occur, and with the frog also there is much more regeneration if this is done.

The bleaching of visual purple in the light and its subsequent regeneration in the dark give us a physical basis for the phenomena of dark adaptation, since by photochemical reasoning we can say that, in order to produce a minimal stimulus, a constant amount of visual purple must be broken down, and the more visual purple there is, the less will be the illumination necessary to produce this amount of breakdown. This simple theory can be stated thus: For a threshold stimulus, ILLUMINATION × CONCENTRATION OF VISUAL PURPLE = A CONSTANT, provided the density of visual purple is small. It must be emphasized that in the orthodox statement of the duplicity theory, visual purple is responsible only for vision at low illuminations, both when a steady state has been reached and during the later part of dark adaptation.

Criticism of the Orthodox Statement of the Duplicity Theory

Although the duplicity theory is the greatest generalization in sensory physiology we shall render it a disservice by giving it nothing but uncritical attachment. Some points of criticism are given in the following sections; others have been published elsewhere (Lythgoe, 1938*a*).

The fate of the rods and visual purple at higher illuminations.—In the investigation of a visual function over a wide range of illuminations there is, as mentioned earlier, one relation between function and illumination for low, and another for higher illuminations. The change occurs at about 0·1 m.c., an illumination approximately equal to that given by a $\frac{3}{4}$ full moon on the earth. By the use of light of different wave-length, by observations made with the peripheral retina and by correlating the habit of a species with its retinal histology, we know that the rods are mainly responsible for vision below 0·1 m.c., and the cones for higher illuminations. We do not know whether vision above and

below this illumination is wholly due to the cones and rods respectively. Our first concern is with the fate of the rods at high illuminations. In curves relating visual function and illumination we can find no trace of a scotopic component at the higher illuminations, and, if the term " rod vision " is synonymous with " scotopic vision," we should say that the rods are playing no part in the visual processes. On general grounds this is surprising, since rods are far more numerous than cones both in number and in the area of retina occupied, and if the rods play no part in vision above 0·1 m.c. it means that they are functionless over the greater part of the scale of illuminations met by the eye (Parsons, 1927).

This objection acquires greater force when we consider the visual purple content of eyes which have been subjected to illuminations above 0·1 m.c. for a considerable time. Even with albino rats placed in an illumination of 2,000 m.c. for a long period, the visual purple is not quite completely bleached (Tansley, 1931). We should expect to find visual purple at much higher illuminations in pigmented eyes. I have exposed Hungarian frogs to the illumination of a lightly-clouded midsummer sky (say 50,000 m.c.) for 40 min. At the end of the period only $\frac{3}{4}$ of the visual purple had been bleached. Since in frogs visual purple may be protected from the light by migrating pigment epithelium, Granit's experiments on cats probably give a truer picture of what is happening in man (Granit, Munsterhjelm and Zewi, 1939). The experiments showed, primarily, that during dark adaptation the electrical response of the retina is very small if the concentration of visual purple is less than 50 or 60 per cent. of its final value, but they also demonstrated conclusively that there must be considerable quantities of visual purple in eyes which have been exposed to illuminations some 10,000 times 0·1 m.c. If the rods and their visual purple play no part at these higher illuminations we must conclude with Granit that the visual purple which is present, although bleachable by light, can produce no stimulus to the optic nerve, and, therefore, no sensation of light.

Complicating factors in the explanation of early dark adaptation. —The terms " photopic and scotopic vision " are purely descriptive and apply, first, to the distinctive phenomena of vision at high and low illuminations respectively. Secondly, the terms apply to the early and late parts of the curve depicting the course of dark adaptation. Although there is little doubt that the adaptation of the cones is mainly responsible for the early or photopic part of the curve, there is some evidence that another mechanism is playing a part. This second mechanism may be

that of scotopic vision or it may be a phenomenon of nervous adaptation, which will be considered later.

As a first approximation it can be said that for photopic vision, visual acuity varies as the logarithm of the illumination. It follows that during the early stage of dark adaptation this relation should also hold if, as is usually stated, vision is then entirely photopic. Recently Dr. A. J. Marshall and I have conducted a series of experiments to test this point (unpublished observations). The observer was given a standard period of light adaptation, and the minimum illumination needed to see a large grating test-object was measured at different times of dark adaptation, the size of grating remaining constant. The grating size was then changed and the experiment repeated. A series of curves relating minimum illumination and time in the dark was thus obtained, one curve for each size of grating. By taking a cross-section at any one time it was possible to construct a second graph, relating visual acuity and illumination for any desired time. The graph so made closely resembles Fig. 3 which was obtained by Hecht and Shlaer (1936). The descriptions are not comparable, since fusion frequency is replaced by visual acuity, and the various traces for decreasing wave-length are replaced by traces for increasing time in the dark. A further difference is that, even for time 60 sec. after the onset of dark adaptation, the derived curve did not show the simple logarithmic relation between grating size and illumination, and the deviation became progressively greater until the final curve obtained after some 15 min. of dark adaptation. The conclusion to be drawn from this experiment is that scotopic vision, or some other phenomenon, is playing a part in early dark adaptation which is usually thought to be exclusively photopic.

The lower limit of photopic vision.—We have seen that the change-over from scotopic to photopic vision occurs at an illumination of about 0·1 m.c. When, however, observations are made with a small centrally fixated field whose image falls on central cones only, it can be shown that the mediating mechanism of photopic vision is sensitive to an illumination as low as 0·0034 m.c. (Hecht, 1921, Sharpley, 1936). Without making any reference to rods and cones, it is clear from an ordinary dark adaptation graph that the first loop of the curve, which is associated with photopic vision, is maintained to an illumination as low as 0·004 m.c. (The kink is at a higher illumination in Fig. 1. The lower value has been found by several authors.) When observations are being made under a steady illumination between 0·004 and 0·1 m.c., there should be enough light to stimulate the photopic mechanism, but scotopic vision seems to be

dominant and there is no trace of a photopic component. Photopic vision seems to become dominant quite suddenly when the illumination is raised above 0·1 m.c., thereafter revealing no scotopic component.

An argument similar to that used in the last section shows that there is probably more than one receptive mechanism in action at illuminations just below 0·1 m.c., that is to say, at illuminations which are generally supposed to be just insufficient to stimulate

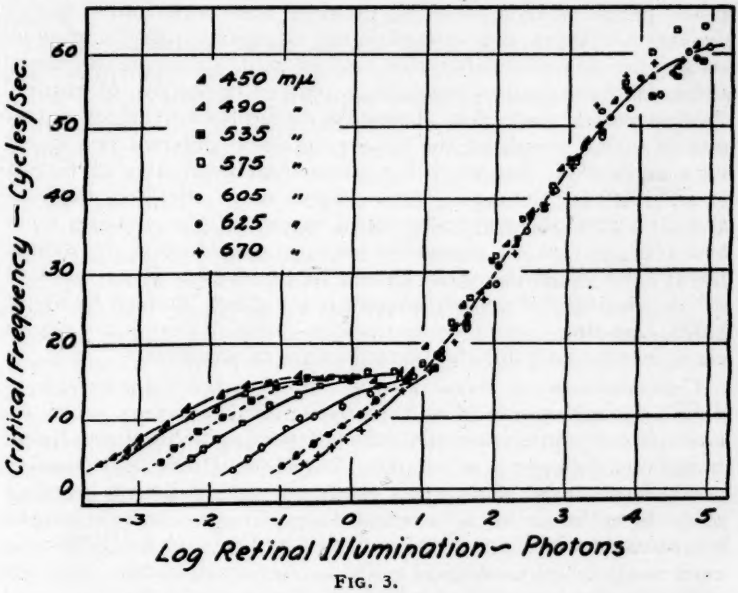

FIG. 3.

The relation between the critical frequency of flicker and illumination expressed in terms of photons.
(A perfectly diffusing white screen illuminated by 3 m.c. and seen through 1 sq. mm. of pupil gives a retinal illumination of about 1 photon.) (Hecht and Shlaer, 1936.)

the cones. This is shown by the experiments of Hecht and Shlaer (1936), where the critical frequency of flicker is plotted against illumination for a number of wave-lengths (Fig. 3). A critical fusion frequency of about 15·6 was obtained at a number of wave-lengths (450, 490, 535, 575 mμ) with a retinal illumination of about 1 photon (that the retinal illumination is the same for all wave-lengths is not significant). If only one receiving mechanism were at work we should expect to find that the values of the fusion frequency would be again equal to one another if the retinal

illumination were reduced to one hundredth. The values were, in fact, about 13·2, 11·5, 10·0 and 7·0 for the 4 wave-lengths respectively.

Concentration of visual purple and threshold stimulation.—Very serious difficulties arise when we attempt a quantitative interpretation of the decrease in the light threshold during dark adaptation in terms of the increasing concentration of visual purple. We saw earlier that for a threshold stimulation we should expect the product of the illumination and the concentration of visual purple to be a constant, provided the concentration is not too large. We must also confine our attention to the increase in sensitivity occurring after the first 10 min. or so in the dark, since visual purple does not seem to play an important rôle during the first part of the curve. There are no figures for the concentration of visual purple in the human retina at different periods of dark adaptation, but we will assume that the figures of Granit *et al.* (1939) for the cat are applicable to man. Between the 10th and 30th min. the concentration of visual purple increases from 57 to 95 per cent. of its final value, and according to the simple theory, the visual threshold should be divided by about one half (57/95) during this period, whereas it is, in fact, divided by about 1,000. In other words the increase in concentration is not nearly enough to account for the great increase in sensitivity.

Concentration of visual purple and threshold stimulation in deficiency of vitamin A.—A further difficulty occurs when we attempt a quantitative explanation of the night blindness found in serious deficiency of vitamin A. Holm (1925) and Tansley (1931) have shown that in this condition there is both a lowering of the light threshold and a retarded regeneration of visual purple, a conclusion which has been confirmed by Charpentier (1936) who used an electro-physiological method. At first sight this illustrates strikingly the essential part played in scotopic vision by visual purple, but quantitatively there is a serious discrepancy. Tansley's rats had been kept without vitamin A for so long that their weights had begun to fall. Man with an equal degree of deficiency would have had a light threshold about 1,000 times normal, and we should expect the concentration of visual purple, both in man and in Tansley's rats, to be about 1/1,000 normal. This means that no visual purple could have been detected by the method of estimation used, whereas there were appreciable quantities in all experiments but one.

Too much emphasis cannot be laid on this argument, however, since deficiency of vitamin A leads to other retinal changes, such as a progressive atrophy beginning with the outer limbs of the rods (Tansley, 1933, Johnson, 1939) and a degeneration of the

nervous elements having its principal effect on the ganglion cells (Mellanby, 1934). The degeneration of the rods may be connected with the paucity of visual purple, since both Granit (1938) and I (1938) think it probable that visual purple is related to the surface of the rod, whilst Schmidt (1938) has put forward evidence that visual purple forms part of the structural framework of the rods. If this view is correct it is not difficult to see that deficiency of vitamin A would produce poor night vision, both by reducing the concentration of visual purple and by damaging its seat of action.

Dark adaptation curves do not retrace one another's paths.—The bleaching and subsequent regeneration of visual purple are more complicated than a simple chemical reaction performed in a test-tube. Let us suppose that we have two test-tubes each containing an equal concentration of a substance which is decomposed by light into two or more substances, or suffers some tautomeric change in the light. Let us further assume that all the breakdown products are stable and recombine in the dark to form the original light-sensitive substance. If the original substances in the test-tubes are bleached, one more than the other, then the recombination in the test-tube which has suffered the greater breakdown will proceed until it has reached the state of breakdown originally found in the less bleached test-tube. If the retinal rods are no more than test-tubes full of visual purple, the bleaching and subsequent regeneration of this substance should behave in the way just described. Assuming both that the final concentration of visual purple is always the same in a given retina, and that the breakdown products are not lost, then regeneration from any given stage of breakdown should always follow the same course, however that stage had been reached. Furthermore, if the same visual threshold is always associated with a given concentration of visual purple, then one curve could describe the course of dark adaptation under all conditions. The same threshold and, presumably, the same concentration of visual purple might be attained at different times in two experiments on an observer, but after equality had been reached the two curves should follow the same course. It has been shown experimentally that this is not so. It is also known that a short flash of light, such as is produced by striking a match in the dark, has only a transitory effect on the process of dark adaptation.

General Conclusions.—Our conclusion, both from a study of the course of dark adaptation and of the nature of the curves relating visual function to illumination, is that the sharp division into photopic and scotopic components does not correspond to a sharp division into cone and rod function. The cones can function at

a lower illumination than is usually stated and they are probably in action for part of the curve conventionally marked scotopic. Similarly, certain phenomena suggest that the rods may play some part in photopic vision, although we shall see later that a nervous change is probably also active in the early stages of dark adaptation. If this view is accepted we shall be entitled to retain the terms " photopic " and " scotopic vision," but we must make it clear that they do not refer to vision which is due exclusively to the cones or to the rods, respectively.

There are still more serious difficulties in the quantitative interpretation of the phenomena of scotopic vision in terms of visual purple. The aim of the rest of this article will be to examine some possible causes of our difficulties.

Regeneration of Visual Purple

According to Kühne (1879) and Hecht (1919-20), visual purple is bleached by light, but simultaneously it is regenerated from its breakdown products, although at a rate which may not be the same as the rate of bleaching. In Hecht's original analysis the breakdown of the light-sensitive substance was directly proportional to its concentration and to the light intensity falling on it. Regeneration was said to occur from two breakdown products and to conform to the equation for a reaction of the second order. The derived equation satisfactorily explained a number of visual relations, such as that found for the discrimination of brightness differences at various illuminations, but it was not satisfactory when given the crucial test of explaining the rate of dark adaptation. It is possible, however, that the assumption of other types of reaction kinetics would have done equally well over a limited range of illuminations. Wright (1935, 1936) found that the hypothesis was inadequate to explain the perception of brightness difference when high illuminations were used. Furthermore, although the rate of regeneration of visual purple may follow faithfully the equation for one of the commoner types of chemical change, it does not follow that what we are observing is primarily determined by a chemical reaction. Physical factors, such as the diffusion of metabolites, may set the pace, whilst the chemical change may occur only when the ground has been prepared for it.

The increase in sensitivity during dark adaptation does not follow an equation either of the first or second order, and from this point of view there are few grounds for thinking that the underlying chemical change is unimolecular or bimolecular. Tansley's (1931) figures, however, did hold out some hope that the increase in the concentration of visual purple with time in the dark might one day be expressed in a simple formula. Granit

and his colleagues have shown, however, that under most, if not all conditions, there is a delay in the regeneration of visual purple. Although this observation may explain the delayed adaptation of the scotopic component of dark adaptation, it adds considerably to the difficulty of a biochemical explanation of regeneration.

If either the increase in sensitivity or the increase in concentration of visual purple during dark adaptation could be adequately described by an equation representing one of the well-known types of chemical reaction, we should have a clue to the nature of the regenerative process. Since no such equation has been found we must do what should have been done before, namely, first study the chemical processes themselves, and secondly explain the time course of the increased sensitivity during dark adaptation.

Course of bleaching of visual purple.—Kühne gave the first account of the colour changes observable when visual purple is bleached by light. Subsequent quantitative work has confirmed the accuracy of his observation. Kühne said that visual purple is bleached by light to form visual yellow, and that the latter subsequently breaks down to visual white. We see at once that bleaching occurs in at least two stages, and that regeneration might be possible from either. Kühne also recognized other colour changes, but he thought they were produced by mixtures of visual purple and visual yellow, and he did not assume the existence of an intermediate link to explain them. My own view, based on a study of the absorption curves at different stages of bleaching, is that light acting on visual purple first produces a distinct chemical compound which I call " transient orange." The latter is very unstable and decomposes thermally in the dark to form " indicator yellow " which, in its turn, forms a colourless compound, especially in weakly acid solutions (Lythgoe, 1937). When Kühne writes of " visual yellow " one can tell only by the context whether he means " transient orange " or " indicator yellow," but it is usually the latter, since special precautions have to be taken to demonstrate the presence of the former substance. My reasons for dropping the term " visual yellow " are, first, that it would be used to describe two substances without any indication of which was meant. Secondly, the term implies that the substance forms a direct link in the chain of events leading to a visual sensation. This is no longer believed. The term " indicator yellow " is used to describe the most obvious character of the chemical substance in question, namely, that it is yellow and that its yellowness changes with acidity (Chase, 1936, Lythgoe, 1937). Transient orange is an unstable substance which always appears orange and whose absorption curve is very little affected by acidity (Lythgoe and Quilliam, 1938*b*). In this I differ from Wald (1938). I have been unable to find any other intermediate stage.

Chemistry of visual purple and its products of bleaching.—It now seems clear that visual purple is made up of a chromophore attached to a protein. This chromophore, or its bond with the protein, is responsible for the characteristic absorption curve. Chemically it is probably a carotenoid which is closely related to vitamin A. If this is so, absence of the vitamin will probably lead to a shortage of visual purple in the retina. The general nature of the chemical changes involved in bleaching have been worked out by Wald (1936). Light causes a loosening of the bond between the protein and its chromophore, so rendering the latter more freely soluble in organic solvents. The substance extracted is called retinene. It behaves like a carotenoid compound, but nothing is known of its structural chemistry. Wald originally seems to have thought that light could split off the chromophore from the protein, and that visual yellow (indicator yellow) was retinene itself. Recently he seems to have modified this view (1938). There are some grounds for thinking that indicator yellow is itself a conjugated protein. We are justified in saying no more than that light weakens the chemical link which is responsible for the characteristic absorption curve of visual purple. It is possible that the essential chemical change is one of hydrolysis (Lythgoe and Quilliam, 1938*a*, Dartnall, Goodeve and Lythgoe, 1938).

The changes just described can be demonstrated either in solution or in the whole retina, but when bleaching has occurred in the retina itself there is a further chemical change. It is found that if the whole retinae are left for some time after bleaching, organic solvents now extract vitamin A itself, indicating that this substance has been formed from retinene. Krause and Sidwell (1938) deny that this is so, finding on the contrary that there is less vitamin A after bleaching than before. If true, this means that in their experiments vitamin A was continuously destroyed, and, since this would apply both to the original and to any newly-formed vitamin A, all we can conclude from their experiments is that this substance is more rapidly destroyed than it is formed, if it is formed at all.

Caution is needed in the interpretation of experiments on solutions of visual purple since these usually contain large quantities of impurities extracted simultaneously from the retina. This criticism applies *a fortiori* to experiments on whole retinae.

A general criticism of Wald's chemical explanation of the bleaching of visual purple and of its regeneration, is that there is no place in it for the action of the carotenoid, xanthophyll, which is known to be present in large quantities in some retinae.

Precursors of regenerated visual purple.—Wald believes that

visual purple can be regenerated from the vitamin A stage in its breakdown. This we can readily understand if at this stage both vitamin A and the protein are present. In deficiency of the vitamin we presumably have the protein alone, and visual purple can be reformed if vitamin A is supplied. Visual purple can certainly be regenerated from solutions of indicator yellow (Ewald and Kühne, 1878, Hecht, Chase, Shlaer and Haig, 1936, Lythgoe, 1937). It is probable that some of the regeneration in the intact retina is from indicator yellow (Boll, 1876, Kühne, 1878, 1879, 1882, and many others). Regeneration from this stage in the breakdown is explained by Wald (1936) as a recombination of protein and retinene, or, according to my reading of his later writings (1938), as a strengthening of the bond between the protein and the chromophore.

Transient orange is an intermediate link in the formation of indicator yellow from visual purple, and recently I have found that under certain conditions regeneration can also occur from this stage in solution. The regenerated substance, although sensitive to light, is slightly different from the original visual purple.

In vitro visual purple can be regenerated from any one of its breakdown products, namely, from transient orange, indicator yellow or visual white. We do not yet know which mechanism normally occurs *in vivo*. We are suffering from an *embarras de choix*. Many workers have shown that the course of dark adaptation varies in kind as well as in degree with the conditions of the previous exposure to light. Wald and Clark (1937) have suggested that the stage of breakdown reached depends on the duration of light adaptation, and that the differences in dark adaptation depend on the rate of regeneration from the various stages. Caution is necessary in accepting this explanation in view of the discrepancy between the concentration of visual purple in the retina and the sensation evoked. Furthermore, the explanation does not take into account the acceleration of regeneration produced by irradiation of the breakdown products. This remarkable phenomenon will now be described.

Factors influencing regeneration (*a*) *Light.*—It is thought that in the intact animal, regeneration is constantly occurring during the whole time that light is falling on the retina (Kühne, 1879, Cobb, 1916, Hecht, 1918, etc.). This we should expect for that part of the regenerative process which is chemical and obeys the law of mass action. Bauer's (1911) experiments showed, however, that the concentration of visual purple depends largely on the previous exposure to light, being *greater* in animals which had been irradiated for the longer period. These experiments have

been put on a firm quantitative basis by Zewi (1939), and it is now clear that previous exposure of the whole retina to light acts as a stimulus to regeneration. Under many conditions, moreover, visual purple does not begin to regenerate immediately the adapting light is switched off. This delay is absent in those animals pre-exposed to light of high intensity or for a long duration. If an observer has been exposed to bright sunlight during the lunch hour the later stage of dark adaptation in all the afternoon runs is retarded, but the early stage of adaptation is found to be quicker (Dr. A. J. Marshall, unpublished work). The pre-exposure to bright light might have caused an accelerated regeneration of visual purple (especially the early phase), and so quickened initial adaptation.

Chase (1937) found that if a solution of visual yellow (indicator yellow) was irradiated strongly there was some regeneration of visual purple which would not otherwise have occurred. I have confirmed this observation, and find that regeneration also occurs under certain conditions from the intermediate transient orange, provided it has been strongly illuminated. It is surprising that the absorption curve and other properties of the regenerated substance are not quite the same as those of the parent visual purple. One cannot help wondering whether the photosensitive substances responsible for day vision are formed in this way: the bright light may be the essential agent in the preparation of the visual purple for the reception of high illuminations.

Factors influencing regeneration (*b*) *The healthy retina.*—Although irradiation can stimulate the regeneration of visual purple in the frog's retina it does not follow that it is an essential factor without which there can be no regeneration. It is certainly not the only factor in regeneration. Tansley (1933) has shown that regeneration is possible in tissue culture, but only in a healthy retina. Zewi has shown conclusively that in the whole retina, regeneration depends on the oxygen supply. Regeneration is greatest when the retina is in contact with a choroid, whose blood circulation is intact, and is least when the retina is isolated, especially if it is put into an atmosphere of nitrogen. According to McFarland and Evans (1939), anoxaemia slows the process of dark adaptation. Phillips (1939) has found that dark adaptation is much slower in old people. Both these observations show that something more than a simple reunion of breakdown products forms the chemical basis of dark adaptation.

It seems possible that we have two methods for the regeneration of visual purple. Regeneration from its breakdown products almost certainly involves the addition of energy to the system, and this energy can be provided either by the absorption of light or by a chemical process needing oxygen.

Possible Nervous Changes during Dark Adaptation

We cannot seriously doubt that the basic physiological change in dark adaptation is a reaction in which visual purple is regenerated, but it is questionable whether this is the only change. There are reasons for thinking that nervous influences are also at work.

Most of what we have said so far has been exclusively concerned with adaptation to low illuminations after exposure to high ones. It is now being realized, however, that dark adaptation is only a special case of a fundamental change which is always occurring when the eyes are being used. The term adaptation is now used to mean the adjustment of the retinal mechanism to the general prevailing brightness of objects in the field of view. By " adaptation " we now mean either the process of adjustment or the final " stationary state " (Hecht) after adjustment is complete. In my opinion something has been learnt of the special case of dark adaptation by studying adaptation in general. Let us imagine that an observer is seated inside a whitened cube, the illumination on whose walls is gradually reduced. The observer's eyes are then being adapted to progressively lower brightnesses. There is a small opening in the cube which is normally closed, but can be opened to afford a short view of some visual test-object which may not be illuminated to the same extent as the walls of the cube. The observer will be performing a visual task at one illumination with eyes adapted to another. Complete darkening of the walls of the cube gives a special case in a wide range of conditions. It has been found that the state of adaptation of the eye, as determined by the brightness of the cube, has a profound influence on the performance of a visual task seen through the hole.

Fig. 2B shows that the perception of flicker is very susceptible to changes in adaptation (Lythgoe and Tansley, 1929*a*). Starting with a high brightness in the cube one first measures the fusion frequency for a flickering light of a slightly lower brightness. Keeping the brightness of the test-patch constant, the brightness of the walls of the cube is progressively reduced until they are equally bright and later less bright than the test-patch. For photopic vision the perception of flicker is best when the test-patch is of the same brightness as the walls of the cube, or, in other words, when the eyes are adapted to the same brightness as that at which they are required to make their judgment. (Readings where the cube was brighter than the test-patch are not recorded on the graph.) The eyes must be allowed time in order to become adapted to each new illumination: in the same way it takes time for the eyes to adapt to complete darkness. Using as a criterion the perception of differences of brightness, Craik (1938) found

that visual perception is at its best when the eyes have been pre-adapted to the illumination at which they will subsequently make their judgment. This appears to be a general law for photopic vision. For scotopic vision, on the other hand, the most favourable condition seems to be dark adaptation, that is when the walls of the cube are completely dark. This is shown by experiments on flicker (Lythgoe and Tansley), to some extent by experiments on the difference threshold (Craik), and by the fact that the perception of small feebly illuminated objects improves during the course of dark adaptation.

The changes in the fusion frequency of flicker during the course of dark adaptation (Fig. 2A) are similar to those found when the adaptation of the eye is changed slowly and progressively by lowering the brightness of the cube's walls (Fig. 2B). Sometimes, as in the measurement of visual acuity, the change during the course of dark adaptation is both too small and too rapid to be measured accurately by the usual method, but by taking readings at progressively lower brightnesses of the cube's walls we can virtually slow the process of dark adaptation and study its phenomena at leisure (Lythgoe, 1932). In much the same way small cinematograph projectors can be stopped to allow a closer view of any one frame.

The finer visual judgments during dark adaptation.—The increasing sensitivity of the eye during dark adaptation is shown not only by the lowering of the light threshold but also by the progressive improvement in performance of visual tasks needing only low illuminations, namely, those calling for only coarse judgments. The finer visual judgments, for which higher illuminations are necessary, behave otherwise and, as we have seen, show a progressive deterioration during dark adaptation. If one adopts a purely chemical outlook on the stimulation of the retina by light, one would expect visual performance to be at its highest when the image of the test object falls on a part of the retina containing a maximum quantity of visual purple or other light-sensitive substance, and not on a retina which had been partially bleached by lighted surrounds.

Nervous interaction.—Adrian has proved that nervous interaction can occur in the retina (Adrian and Matthews, 1928), although its presence had been suspected for a long time. Nervous interaction between those parts of the retina receiving the image of the lighted cube and the fovea, affords the most likely explanation of the influence of the surrounding field on central vision. Recent experiments by Schouten (1937), and Schouten and Ornstein (1939), even make such an explanation probable. We shall see that it is also probable that a nervous change plays a part in the ordinary process of adaptation to darkness. By the

method of binocular matching (*e.g.*, Wright) Schouten made a quantitative record of the apparent brightness of a patch of light, when there was exhibited near it another source of light, which for convenience we will call the glaring source. Everyday observation has taught us that " When the moon shone, we did not see the candle . . . . so doth the greater glory dim the less " (Merchant of Venice). Experiment is necessary, however, to demonstrate the time course of the phenomenon and the great extent to which the apparent brightness of the test-light is depressed by a neighbouring source of glare. The reduction in the apparent brightness took place in about 1/10 sec., and the value so reached was maintained for the duration of the experiment. This process of *a*-adaptation, as it is called, cannot possibly be photochemical in origin, both because it takes effect so quickly, and because the effect is exercised at a distance. Much the most likely explanation is that glare produces its effect by some variety of nervous interaction. Our concern, however, is with the recovery from the glare which resembles part of the process of dark adaptation. We have seen that glare reduces the apparent brightness of the test-patch. Removal of the glare causes a restoration of the original brightness, but this process takes considerably longer than 1/10 sec. Since the original suppression of brightness was due to nervous interaction, we may assume that some part of the recovery from the glare is also due to a nervous change. The uniformly lighted field of view, such as is used for the preliminary light adaptation in experiments on dark adaptation, may be regarded as made up of a multitude of contiguous glare sources. Each one of these will produce an effect on the test-patch and some part at least of the recovery from the effects of high illumination, or, as we call it, the process of dark adaptation, will have a nervous component.

By means of the Pulfrich rotating pendulum I have shown (Lythgoe, 1938*b*) that a glaring source of light *shortens* the latent period of vision for another light exhibited nearby. A uniformly lighted field of vision has the same effect. The phenomenon can be explained best by assuming that some sort of nervous interaction is taking place in the retina. It is mentioned here to show that changes in the adaptation of the eye produce many and varied effects.

Dark adaptation as a combination of both chemical and nervous changes.—I look on dark adaptation as a complex phenomenon in which the eye both becomes more sensitive to light and performs better the grosser types of visual judgment where the recognition of the presence or absence of light is the most important factor. Simultaneously the eye becomes progressively less able to perform the finer visual tasks. The lowering of the light threshold is due mainly to the regeneration of visual purple: the increased sensitivity of the photopic mechanism (Hecht, 1921, Wright, 1937)

is probably due to a similar change. The decreasing ability to perform the finer visual judgments may be due to synaptic rearrangements, which make the retinal elements more capable of initiating an impulse, but less capable of performing fine judgments. The sort of change I envisage is shown in Fig. 4. During complete light adaptation let us consider a number of visual elements, each one of which is served by a separate nerve fibre. During dark adaptation each fibre serves several elements

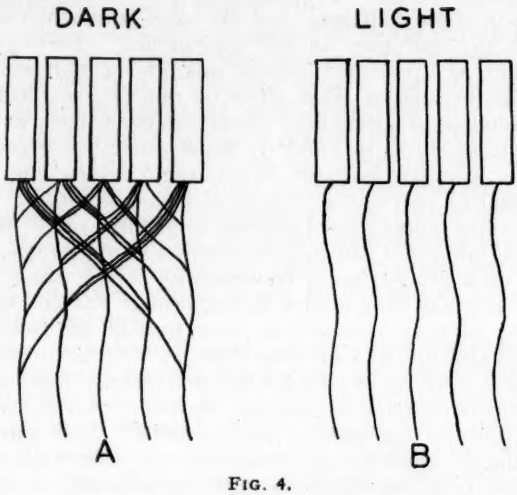

FIG. 4.

Illustrates the suggestion made in the text that there is more overlapping of the nerve supply to the rods and cones during dark adaptation than during light adaptation.

by a spread of its synaptic connections. This would reduce the fineness of discrimination but it would probably improve the retinal sensitivity to light since it is known that although an image of a certain brightness falling on one rod (or cone) may produce no sensation of light, a larger image of the same brightness falling on two or more rods (or cones), may do so. Such an integration may be possible only when several rods connect with one nerve fibre (Fig. 4A) and not in the light-adapted arrangement (Fig. 4B).

If we regard the process of dark adaptation as one in which, *inter alia*, the rods or cones acquire progressively more nerve fibres in common, we must regard the process of light adaptation as one in which the rods or cones become progressively more segregated in their nervous connections. If this were so it would explain

the improvement in visual acuity with illumination, a phenomenon which has proved very difficult to explain. One would explain the improvement in visual acuity produced by a surrounding visual field of suitable brightness by saying that it induces the discrete arrangement of cones and nerve fibres at the fovea.

Although in this way we can explain the change in performance of those visual tasks which are dependent on the spatial selectivity of their central representation, we have difficulty in explaining the change in the discrimination of brightness differences with adaptation. One cannot say whether it would be explained by the rearrangement of nerve fibres shown in Fig. 4. If the cones to the left hand of Fig. 4A or 4B were more highly illuminated than those to the right hand, then the corresponding nerve fibres on the left would discharge more rapidly than those on the right. The transition from fast to slow would be more abrupt in the fibres from the discretely represented cones in Fig. 4B which represents light adaptation, than in Fig. 4A which represents dark adaptation. A blunt edge to the comparison fields is known to affect adversely the discrimination of brightness differences, but it is questionable whether the apparently blunt edge which would be seen in Fig. 4A would explain the poor discrimination of brightness by the dark adapted eye.

Many of the difficulties in accepting a purely photochemical explanation of the phenomena of dark adaptation have been taken from the work of Granit, Holmberg and Zewi (1938*a*, *b*). These authors have shown that the electrical response of the retina is not solely dependent on the concentration of visual purple. The virtual absence of an electrical response may occur in spite of a relatively high concentration of visual purple. These authors suggest that part of the visual purple may be inactive physiologically, although capable of being bleached by light. I have a bias against any view which assumes that visual purple is being bleached to no purpose. Although it may not be so for other physiological activities, the sense organs usually work most economically. I prefer to think of each molecule of visual purple as being a potential initiator of a nerve impulse when broken down by light, but that the final electrical response of the retina and optic nerve is determined by the existing synaptic arrangements. I feel that Granit is right, however, to stress the view that visual purple is closely connected with a surface in the rods. If the rod is a nerve cell one is much inclined to the view that when light bleaches a molecule of visual purple lying in the surface, an electrical disturbance is initiated at that spot which may be able to start an impulse in an optic nerve fibre.

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SPHERO-CYLINDRICAL CONTACT LENSES—A PRELIMINARY NOTE

BY

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SOME two years ago, one of us was examining a patient who had been fitted with contact lenses and was disappointed to find that visual acuity with the contact lens was less good than with ordinary glasses. The spherical element was checked by the duochrome test and appeared to be right, so retinoscopy was performed and it was discovered that there was some residual astigmatism. When the correcting cylinder was placed in a trial frame in front of the eye wearing the contact lens an immediate improvement in visual acuity became manifest. It was then suggested that an endeavour should be made to grind the cylindrical correction on to the contact lens, but this could not be done with the machinery available at that time. Recent improvements in the technique of contact lens manufacture have now rendered this possible, however, and although only four cases have so far been treated in this manner, we felt that the results were sufficiently good to warrant the issuing of a preliminary report.

Before coming to the clinical aspect of this work it is not out of place to consider the theoretical side. When contact lenses were first produced, it was assumed that a cylindrical correction was unnecessary because astigmatism was entirely due to aberrations in the anterior surface of the cornea, and was therefore neutralised by covering the aspherical surface with a thin layer of saline and a spherical shell of glass. Perhaps this was making

a virtue of necessity because the type of lens then in vogue had a spherical surface in contact with the bulbar conjunctiva, and theoretically there was therefore nothing to prevent the lens rotating. In practice, however, rotation does not occur, but there is the difficulty that the lens must be inserted in exactly the right position and this would necessitate the making of a fairly conspicuous mark on it which the patient could see. With a contact lens whose scleral surface has been ground so as accurately to fit the ball of the eye, this is not the case, because even if the lens is inserted in a faulty position it tends in a short time to settle down into the position which gives it the closest fit to the eyeball. A good scleral fit, therefore, is of the utmost importance.

The existence of astigmatism which is not due to aberrations in the curvature of the anterior surface of the cornea has been recognised for a long time.

Theoretically, such astigmatism may be due to:—

(1) Aberrations in curvature of the posterior surface of the cornea.

(2) Aberrations in curvature of the anterior and/or posterior surface of the lens.

(3) Radial errors due to the centre of the lens not coinciding with the optical axis of the eye.

(4) Variations in the refractive index of the lens in different meridians.

(5) Tilting of the lens.

It is obvious that none of these can be corrected by a contact lens with spherical corneal surfaces, and it is not surprising therefore that cases arise in which visual acuity is improved by the addition of a cylindrical element to the contact lens.

It is even conceivable that cases which, with ordinary glasses appear to have a spherical error, may when fitted with a contact lens be found to have some residual astigmatism, since it has been stated that most normal eyes show slight corneal astigmatism, but that nature has provided a compensating lenticular defect. If, in such a case, the corneal astigmatism is abolished, the compensating lenticular astigmatism will become manifest, and require correction. The theoretical implications of this are obvious and a considerable amount of work can still be done on the subject, despite the fact that many papers have already been written on the disparity between keratometer readings and the total refraction of the eye.

Turning now to the practical side of the question, a cylindrical correction has not heretofore been ground on a contact lens because it was not possible to combine this with the curvature required on the anterior surface. It has proved possible, however,

to grind such a correction on the posterior (concave) surface of the lens; but here an interesting complication arises, because we are dealing with the difference between the index of refraction of glass and of the saline which fills the space between the anterior surface of the cornea and the posterior surface of the contact lens.

It follows therefore that, as the difference between the refractive indices of glass and saline is much less than the difference between the refractive indices of glass and air, a more powerful cylinder will be required. Calculation has shown that the increase in strength is approximately 2·5, *i.e.*, a cylinder of 2·5 D. ground on the posterior surface of a contact lens will produce the effect of a 1·0 D. cylinder on the anterior surface. By using this method, satisfactory results have been obtained in the following cases, though it is possible that as the result of further investigations they may be improved upon.

S.G.B. male.—Although this patient's error of refraction was only a small one, he was anxious to have contact lenses.

Wearing ordinary spectacles, the right eye with $\frac{\text{sph} - 1{\cdot}0}{\text{cyl} - 0{\cdot}37 \swarrow 5}$ read 6/6

and the left eye with $\frac{\text{sph} - 1{\cdot}62}{\text{cyl} - 0{\cdot}25 \searrow 130}$ read 6/6.

With the usual type of contact lens, each with sph + 2·50 ground on it, vision would not improve to beyond 6/9.*

With the appropriate cylindrical correction ground on to the back of each contact lens, however, each eye could see 6/5. Writing the new contact lens corrections in effective power, they were respectively:—

R.E. $\frac{\text{sph} + 2{\cdot}0}{\text{cyl} + 1{\cdot}0 \rightarrow 180}$ L.E. $\frac{\text{sph} + 2{\cdot}0}{\text{cyl} + 0{\cdot}75 \rightarrow 180}$

The cylinder, as already explained, was actually 2·5 times this amount. (The same convention is adopted in the following three cases.)

* It should perhaps be explained why a patient with a myopic spectacle correction should require for the rectification of his error of refraction a contact glass with an effective power of + 2·50 D. The reason is that in this particular case, the patient's cornea was more curved than the corneal portion of the standard 8 mm. radius contact lens. In consequence of this, without a contact lens, the eye formed an image in front of the retina. When the contact lens was applied, however, the refractive power of the eye was lessened owing to the decreased curvature of its effective anterior surface. In consequence of this, the focus was lengthened and so the image fell behind the retina, thus making the eye hypermetropic and rendering necessary the grinding of a convex lens on to the anterior surface of the contact glass.

O.G. male.—The next was a moderate myope. The spectacle correction which gave 6/5 partly in each eye was:—

R.E. $\frac{\text{sph} - 6{\cdot}0}{\text{cyl} - 0{\cdot}50} \searrow 140$ L.E. $\frac{\text{sph} - 5{\cdot}50}{\text{cyl} - 1{\cdot}0} \swarrow 10$

With a spherical contact lens −3·50 D each eye read 6/6, but with a sphero-cylindrical one

R.E. $\frac{\text{sph} - 3{\cdot}0}{\text{cyl} - 1{\cdot}0} \searrow 100$ L.E. $\frac{\text{sph} - 3{\cdot}0}{\text{cyl} - 1{\cdot}0} \swarrow 80$

each eye could read 6/5.

The third patient, M. R. (male), was referred to the Contact Lens Centre by Mr. Myles Bickerton to whom we are indebted for permission to refer to the case. The right eye with spectacle lens saw 6/5, but the left was amblyopic. With a spherical contact lens +3·0 D each eye, the right would not improve to beyond 6/6, and the amblyopic left eye saw 6/24. With a sphero-cylindrical contact lens $\frac{\text{sph} + 2{\cdot}75}{\text{cyl} + 0{\cdot}75} \swarrow 10$ the right eye saw 6/5.

The fourth case, Mr. O. aged 19 years, was one of conical cornea. This patient was a West Indian. He developed a small amount of myopia about four years ago and this increased steadily. He was diagnosed in Jamaica as early conical cornea and proceeded to England to obtain contact lenses. With spectacles (concave sphero-cylinders) he could read 6/12 with the right eye, and 6/6 partly with the left, but complained that, although he could see the letters, they had double outlines, which worried him all the time. With a spherical contact lens he read 6/9 R.E. and 6/6 partly L.E. practically the same as with his glasses. By adding a 2·5 cylinder to the back of the contact lens of his left eye, he read 6/5 easily, and all the double outlines disappeared. The right eye was not improved, but he stated that he thought the visual acuity in this eye had always been less than in the left.

ANNOTATION

The Leeds Reading Aid for Partially Sighted Children

The Report of the School Medical Officer (Dr. St. Clair Stockwell) for the year ended December 31, 1938, in the City of Leeds, contains many items of importance. To ophthalmologists Appendix B, which deals with the subject of our heading, is of great interest. In an historical retrospect we are told that a class for partially blind (now

termed partially sighted) children was formed at Leeds in 1911 and there were enough children of this type to form two classes. In 1917 reorganisation for the admission of myopes was undertaken and for some years there were two classes for the partially blind and one for myopes. The first class was allowed to use books of good type but the myopes none at all. All writing was done with white chalk or crayon on a black surface. Children, whose sight the ophthalmic surgeon considered "very bad" were placed in classes with blind children and taught on blind lines. The results were not altogether satisfactory. The Report on the education of partially sighted (1934) showed that nearly all these children would have to earn their living in sighted occupations and advised that they should be taught on sighted lines.

A great deal of preliminary work was done by Mr. Lodge and Mr. Harry Lee to improve matters and a device has been perfected for allowing magnification of the print by a glass. The lens used is +3·5 D. plano-sphere, the plane as the under surface. It is made in an oblong block measuring 6½ ins. by 3¼ ins. and has a rigid frame which is attached to the desk. A linear magnification of 2 is thus given, the glass is wide enough to cover the page of most books and the height gives a sufficient length of the page for most readers. A cowl for a lamp is built into the top part of the frame and acts as a reflector.

Research in illumination has shown that an amber tint is best for myopes and a pale flesh tint for other partially sighted children.

A 20-watt bulb gives best illumination for myopes and a 25-watt bulb for partially sighted. "A rheostat control would decide the most suitable intensity for the few abnormal cases."

Twenty-nine myopes have been using the aid for the 12 months covered by the Report.

Regular examination of these are shown:

1 (*a*) Number showing no increase either before or since using 5
(*b*) ,, ,, increase before but none since using - 16
(*c*) ,, ,, increase before and since - - - 4
(N.B.—These children in the first three items of the schedule have been under observation for periods varying from three to nine years.)
(*d*) Number whose progress before using was unknown - 4
2 (*a*) Number showing no increase since using - - - 24
(*b*) ,, ,, increase since using - - - 4
(*c*) Not finally estimated (in Hospital) - - - - 1

The authors of the Report claim that there is no doubt that the aid has been of benefit in education, and the children like the aid.

It is to be hoped that other educational authorities will adopt the Leeds aid and we feel that the device is likely to be of great assistance and that Leeds deserves congratulations for this research and the results.

ABSTRACTS

MISCELLANEOUS

(1) **Pincus, L. (Groningen).—Tumours of the Iris. (Neubildungen der Iris).** *Ophthalmologica*, Vol. XCVII, p. 302, 1939.

(1) In a clinical report with histological findings **Pincus** describes: (1) a spindle-celled sarcoma of the iris that was removed by iridectomy; (2) a ring sarcoma of the iris; (3) a tumour which had originated from the cells of the secondary optic vesicle, probably a genuine glioma in a man of 30 years of age; (4) a new growth in the anterior chamber of the eye, an endothelial proliferation or tumour-like development from congenital remains at the angle of the anterior chamber.

ARNOLD SORSBY.

(2) **Levy-Wolff, Lizzie.—The pathogenesis of retinitis pigmentosa. (Die Pathogenese der retinitis pigmentosa). (Sclerosis pigmentosa chorio-retinalis).** *Acta Ophthal.*, Vol. XVII, p. 192, 1939.

(2) **Levy-Wolff** reviews the more recent findings of unassociated lesions of retinitis pigmentosa and holds that (angio) sclerosis pigmentosa chorio-retinalis would be a more apt name, as she believes that the condition is essentially vascular. Drawing an analogy with amaurotic idiocy, and on the strength of cholesterin analysis of the blood, she suggests that a large group of metabolic disturbances constitute one entity, retinitis pigmentosa being one of the variants.

ARNOLD SORSBY.

(3) **Santoni (Naples).—The metabolism of the retina after interruption of the retinal circulation. (Il ricambio della retina dopo interruzione della circolazione retinica).** *Ann. di Ottal.*, April, 1939.

(3) **Santoni** made a number of experiments on rabbits and rats to show the changes in metabolism after the circulation through the eye was stopped. He found that if the circulation was interrupted for not more than half an hour, the amount of oxygen used was increased; if the interruption was longer, it was diminished. Glycolysis, on the other hand was little affected by stoppage of the blood supply. The author's experiments tend to show that the retina has but little power of re-establishing its functions if the loss of circulation lasts more than a few minutes.

He concludes that if, circulation has been stopped by an embolus,

the chance of recovery is very small. He thinks that the increase of oxygen consumption, which he has noted after a short stoppage of the circulation, is the result of an increased activity of autolytic ferments during the destruction of the retinal cells, and the appearance of fatty acids in this process, bodies which increase the consumption of oxygen.

HAROLD GRIMSDALE.

(4) **Cavallacci (Pisa).—A case showing the Laurence-Biedl syndrome with slight degree of microphthalmos. (Sindrome di Laurence-Biedl con microftalmo bilaterale congenito di lieve grado).** *Arch. di Ottal.*, March, 1939.

(4) This syndrome is a sign of faulty functioning of the region of the hypophysis and diencephalon. In the case under consideration, there was adipose genital dystrophy of the typical Fröhlich form. In addition the eyes were small, the corneae being 10 mm. in the transverse diameter and 9·5 mm. in the vertical.

There were supernumerary digits on the left hand and both feet. Those on the left hand were removed by operation.

Both eyes showed retinitis pigmentosa, with contracted visual fields and some slight diminution of acuity.

Cavallacci raises the question whether all these things are not due to defective development of the prosencephalon, from which both the optic vesicles and the posterior lobe of the hypophysis spring.

HAROLD GRIMSDALE.

(5) **Sala (Palermo).—Ocular changes due to Stovarsol. (Alterazioni oculari da stovarsol sodico).** *Annal. di Ottal.*, March, 1939.

(5) All arsenic compounds are more or less dangerous to the eyes; it has been stated that stovarsol is less dangerous than others and on this account it has been pronounced to be the drug to be selected when it is desired to give arsenic. Some disasters have been recorded even with this compound. Its composition is acetyl-amino-oxyphenylarsenic acid, and it is pentavalent.

Sala wished to examine the toxicity of stovarsol and chose for experiment, the sodium salt; he injected a solution intravenously into rabbits, in some cases with the addition of some quantity of alcohol. After some months of treatment, the animals were killed and the eyes and optic nerves examined; he found evidence of atrophic degeneration of the retina and also of the optic nerve. He concludes that the prolonged administration of stovarsol is not devoid of risk, especially when the patient is in poor condition.

HAROLD GRIMSDALE.

CORRESPONDENCE

IRITIS IN FOWLS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY

DEAR SIRS,—I read with great interest in your Journal the paper on iritis in fowls by McDonagh and Wolft (Vol. XXIII, p. 659). May I add a few remarks concerning the question.

Fowl paralysis was described by J. Marek, Emeritus Professor at the Veterinary High School, Budapest, in 1907. He was able to separate the disease from the beri-beri type of paralysis. This morbid picture was observed for the first time in three Orpingtons and one Langham. His histological investigations have shown the thickening of nerves and plexi. In 1929 the disease was reported in English breeds by Galloway.

In 1932 Warrack and Dalling have pointed out the common symptom of iritis. The most important work on fowl paralysis is that of Seager, director of Poultry Pathological Research Laboratory, Goring, who gave full description of the clinical manifestations and aetiology of the disease.

An admirable account of ocular symptoms was given by Morax and associates in 1934. The histological findings are quite similar to those of McDonagh and Wolff; chronic iridocyclitis, which gives rise to secondary changes (cataract). Corneal oedema was noted. Resemblance to chronic human infections (syphilis, tuberculosis) is emphasized. The term used for "white eyes" is *yeux opaques* or *perlés*.

It must be borne in mind that blindness occurring in this disease may be observed in chickens without any external pathology. In this case changes in the optic pathways, especially chiasma, have to be suspected, similar to those of peripheral nerves, vagus, splanchnic nerve, perivascular infiltration, etc. As to the aetiology, the present view generally accepted is that fowl paralysis is caused by filterable organotropic virus. The same may be said concerning optic changes.

I am, Sirs,

Yours very truly,

STEPHEN DE GRÓSZ, M.D.,
Assistant at the University Eye Clinic, Budapest, Hungary.

BUDAPEST,
November 1, 1939.

REFERENCES

MAREK, J.—*Deutch Tierarzte, Woch.*, Vol. XV, p. 417, 1907.
MORAX, Truche, Vida—*Ann. d'Ocul.* Vol. CLXXI, p. 383, 1937.

NOTES

Death

IT is with great regret that we record the death of Wilfred Trotter, late Serjeant-Surgeon to the King. He was distinguished not only in the realm of neuro-surgery, but also as a profound psychologist.

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Honour

IT is with very great pleasure that we congratulate Mr. H. L. Eason on his recent election as President of the General Medical Council.

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The Herman Knapp Memorial Eye Hospital

THE Herman Knapp Memorial Eye Hospital of 500 West 57th Street, New York City, will be taken over by the Columbia Presbyterian Medical Centre and Columbia University on January 1, 1940. The Eye Hospital in 57th Street will close on that date and the hospital activities and the care of the out-patient department will be continued by the Eye Institute of the Presbyterian Hospital and the Vanderbilt Clinic. The assets and funds of the Knapp Hospital, including the present building, will go to Columbia University to found the Knapp Memorial Foundation in Ophthalmology. The income is to be used for study, post-graduate teaching and fellowships. The Foundation will be directed by a committee consisting of Dr Arnold Knapp, Dr. Phillips Thygeson, Executive Officer of the Department of Ophthalmology of Columbia and Dr. Willard C. Rappleye, Dean of the College of Physicians and Surgeons.

The Herman Knapp Memorial Eye Hospital was founded by the late Dr. Herman Knapp in 1869 and was then known as the New York Ophthalmic and Aural Institute, at 46 East 12th Street. During its first year the institute cared for 1,828 patients in the dispensary and 143 resident patients. In addition, instruction was given to 46 students. *Arch. of Ophthal.*, Vol. XIV, p. 909 (December), 1935.

The institute remained at 46 East 12th Street for 43 years. An addition was built in 1879 to make room for a laboratory which also served as a lecture room. In 1894 the adjoining house, at 44 East 12th Street, was acquired and remodelled as an out-patient department, with optician and drug room facilities and a clinical operating room.

In 1909 Dr. Herman Knapp was succeeded as executive surgeon by his son, Dr. Arnold Knapp. In 1913 the institute was moved to

57th Street and Tenth Avenue; its name was changed to the Herman Knapp Memorial Eye Hospital and the aural department was abandoned. In addition to its regular medical work and post-graduate teaching most of the instruction in eye diseases was given there to the students of the College of Physicians and Surgeons until the college moved to its new site, in 1928.

Though the work of the Hospital in its 57th Street location was most satisfactory, the Trustees of the Hospital have realized for some time that the future of the smaller specialty hospitals in New York is a hazardous one, and after careful consideration came to the conclusion that close association with a large general hospital and a university medical school was essential.

This institution is completing its 70 years; 43 years at 46 East 12th Street, and now 27 years at 500 West 57th Street. In this period 800,000 patients have been treated; 730,000 in the out-patient department without any charge and 70,000 in-patients of whom 76 per cent. were treated free. While the cessation of this hospital in its present location was only decided upon after the most careful deliberation, the Trustees feel that in the coming merger with the Columbia Presbyterian Medical Centre a forward step may be heralded and the original ideas of the founder, Herman Knapp, could be carried out in adequate and permanent form.

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Australian Medical Congress

The Sixth Australian Medical Congress (B.M.A.) which was to have been held in Perth, Western Australia, in September, 1940, under the Presidency of Dr. D. D. Paton, Honorary Ophthalmic Surgeon, Children's Hospital, Perth, has, on account of the international situation, been indefinitely postponed.

FUTURE ARRANGEMENTS

February 3, 1940.—North of England Ophthalmological Society, at Liverpool.

March 16, 1940.—North of England Ophthalmological Society, at Sheffield.

May 4, 1940.—North of England Ophthalmological Society, at Newcastle-on-Tyne.

July 4-6, 1940.—Ophthalmological Congress, at Oxford.